hunger pain. Attractive as this theory of bulimia is, it seems that it must be an infrequent explanation, from negative evidence. In patients with cancer of the cardiac end of the stomach, with rapid, sometimes instantaneous emptying, hunger has not been present. My roentgenologic colleague, Dr. R. W. Mills, tells me he has not seen it in any of some 25 cases of rapid emptying after gastroenterostomy.

It is perhaps going too far to assert that the treatment was the cause of the improvement. Many will think the condition was a neurosis and the improvement a coincidence, perhaps only temporary. However that may be, I think the case has a lesson in showing the advantage of treating local diseases that may cause functional disorders rather than resorting to the narcotics, depressants or other symptomatic remedies still recommended and used in such cases.

The case illustrates another feature of therapeutics. For years medical teachers have emphasized the importance of "treating the patient and not the disease," of ascertaining the complete anatomical and functional condition of the patient and correcting all anomalies. This would seem to mean that all morbid processes should be treated, instead of treating abstract conceptions.

Focal infections, sometimes causing local symptoms, always capable of setting up other and dangerous processes, would seem to indicate careful and exhaustive treatment. And yet we see all kinds of neuroses in patients with infections of the teeth, tonsils, prostates, or appendices, but little or nothing done with the demonstrated foci unless a haphazard effort with vaccines.

A STUDY OF MIGRAINE.

By Mary P. S. Rupert, M.D.,

AND

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An eminent English neurologist has said: "There is no problem that requires more thorough and searching investigation than the origin of headaches, and there is no condition so unimportant that it can be overlooked in any case of headache."

With the above axiom in mind, the work on the study of migraine was undertaken—to find the origin of the disturbance, and if possible to correct it. In a condition so varied, the lines presenting for investigation are indeed numerous, and, we believe, none are too trifling to be passed by.

Migraine is the commonest of all forms of headache. This is hardly disputable by anyone who takes the trouble carefully to investigate the history. On the whole, more women than men are attacked—the proportion about 6 to 4—and social status seems to exert very little influence, as one finds the condition as frequently in the poor as in the idle rich. On the other hand, it is probable that brain workers in consequence of their mode of life are more frequently and severely visited than those whose business is in the open air.

The symptoms begin usually in childhood. In individual cases, however, the condition makes its appearance only in the third or fourth decade. If the onset is given with certainty as late as this, the diagnosis should be made with caution.

There are few ills which depend to the same degree as migraine upon direct homogeneous heredity. Moebius, indeed, found that 90 per cent. of his cases showed direct heredity. This point is of great theoretical and practical interest.

The predisposition to hemicrania must be distinguished from the exciting causes of the outbreaks, and frequently the first attack follows one of the infectious diseases, typhoid, scarlet fever, influenza and the like. Among the actual exciting causes of individual attacks are: emotional disturbances, anger, any mental overexertion and alcohol. Constipation and menstruation play a very important role. It is a fact that disease of the mucous membrane of the nose and accessory cavities and of the ears, as well as irregularities of the eyes, will precipitate attacks. Migraine not uncommonly accompanies a patient throughout life, although with considerable intermissions.

This condition so prevalent among us has been known since the time of Hippocrates, and derives its name from the Greek word, which means hemicrania. It was so named by Galen.

In contradistinction to the word *hemicrania*, the pain is not necessarily so limited, and, indeed, in certain cases of migraine there is no headache at all.

Migraine is not a disease entity, but rather a symptom-complex occurring in various forms and intensity in different people.

In order to provide a background, we shall use a modification of the classification as given by Flateau.

1. MIGRAINUS VULGARIS. This is a frequent form, accompanied by pressure in the head, lassitude, anorexia and nausea. The eye symptoms and actual pain may or may not be present. This type rarely has an aura.

2. OPHTHALMIC TYPE. The symptoms are legion, but the striking and ever-present few which may make the diagnosis for the doctor when seeing the patient are: sensitiveness to light and noises, with scotoma scintillans; exquisite pain in the head, not necessarily limited to a hemicrania, but felt over the entire head,

with aching in the jaws, the nose and the occiput region. This type is accompanied by nausea and vomiting and great prostration during the attack. Paralysis of individual ocular muscles sometimes occurs during the attack, with some resulting ptosis.

3. VASOMOTOR TYPE. This has, if possible, more symptoms to its credit than the ophthalmic variety, and among them may be included: hemicrania, or diffuse pain; angioneurotic edema; abdominal pain; disturbances of peristalsis; cardiac depression; breathlessness; yawning or sneezing, with marked dizziness.

4. PSYCHICAL MIGRAINE. Psychical migraine differs from the vulgaris type in that it has marked psychical disturbances within physiological limits, such as temporary loss of memory, temporary aphasis, hysterical spasm, garrulousness and melancholia.

Pathology has shown no demonstrable lesion; therefore, the cause has been the subject of innumerable speculations. It may be readily understood how such varied symptoms in so many parts of the body lead to varied hypotheses. It would seem to us the theories most worthy of mention are:

The Reflex Theory. This is based upon the idea that the condition is produced by a reflex stimulation of some important organ, such as the uterus, eye, nose, stomach and the like.

The Vasomotor Theory. The vasomotor theory depends upon the vasomotor processes and upon arterial hyperemia. In this theory certain groups of symptoms are explained also by venous or passive congestion, while still others are to be accounted for by weakened functional control. These changes may cause marked vascular contractions or the so-called angiospastic form.

The Central Theory. The central theory would prove the process as primarily in the brain substance, the principal factors being hyperemia or congestion of the brain cortex and meninges. Spritzer suggests that an excessive secretion of the choroid plexus is associated with either a functional or anatomical stenosis (partial or complete) of the foramen of Monro and a subsequent congestion of the brain. Deyl believes a swelling of the hypophysis to be the underlying factor. Quincke holds that an angioneurosis causes increased brain-pressure and Schulke introduces the colloid theory in attempting to account for too much brain substance for the skull capacity. However interesting these points may be they are entirely hypothetical, and careful histological-anatomical examinations have not confirmed them.

The Toxic Theory. The toxic theory presupposes some disturbance of metabolism, and most frequently mentioned is gout in its so-called neuroarthritic form, or a disturbance of the endocrine system, or a combination of both.

ANALYSIS. Consideration of all these views leads to the conclusion that the symptoms of migraine arise from stimulation of certain areas in the central nervous system and that this stimulation may be provoked in many ways: by pressure, by reflex stimulation or by circulation in the blood of certain toxic materials, the nature of which has not been satisfactorily determined.

It is with the last named that our studies have been chiefly concerned. With this factor in view we have tried to find a possible source of poison.

Case No.	Age.	Heredity.	Age at onset.	Frequency of attacks.	Possible contributory factors.
1	38 years	Migraine; carcinoma	20 years	Every three or four weeks	Chronic tonsillitis.
2	24 years	Negative	22 years	Two or three times a week	Astigmatism.
3	56 years	Negative	18 years	Every two or four weeks	Pyorrhea and dental caries; cholelithiasis (operation three years ago); eye trouble.
4	32 years	Syphilis; tuberculosis	18 years	Every two weeks	Artificial menopause.
5	45 years	Negative	20 years	Every ten days	Pyorrhea.
6	48 years	Tuberculosis	25 years	Once a week	Pyorrhea.
7	36 years	Negative	14 years	Once in two weeks	Cervical lymphangitis. pyorrhea.
8	40 years	Carcinoma	33 years	Once a week	Goitre; cholelithiasis (operation one year ago); pyorrhea.
9	33 years	Rheumatism; chorea	26 years	Once or twice a week	Spur in nose.
10	22 years	Tuberculosis	Childhood	Irregular, associated with unusual mental strain	Astigmatism and strabismus.

Case No.	Wassermann.	Blood count.	Routine urinalysis.				Blood-pressure.	
			Sp. gr.	Albumin	Casts.	Indican.	Systolic.	Diastolic
1	Negative	Normal	1021	−	+	+	100	74
2	Negative	Normal	1030	−	−	−	92	62
3	Negative	Normal	1027	−	+	+	176	93
4	Negative	Normal	1011	−	−	−	101	65
5	Negative	Normal	1032	+	−	+	116	72
6	Negative	Normal	1021	+	−	+	120	75
7	Negative	Normal	1012	−	−	−	100	70
8	Negative	Normal	1015	−	−	−	113	69
9	Negative	Normal	1015	+	+	−	113	68
10	Negative	Normal	1013	+	−	−	118	72

In each case a careful study has been made of the gastric contents and the feces. The problem of nitrogen retention was studied by the quantitative estimation of urea in the blood, and the kidney function was investigated by the tests which have been accepted as most dependable, *i. e.*, the phthalein and the Mosenthal test diet.

To eliminate other possible factors and to round out a complete study of the case we have included the Wassermann test, the blood count and routine urinalysis. A careful history has been taken of each patient and a thorough physical examination made in a search for predisposition and for an exciting cause.

In the course of the physical examination we have noted that many cases of migraine run an abnormally high blood-pressure during the attack; therefore, we have made a careful study of this particular phase of the subject, estimating the blood-pressure both during and between the attacks.

It has been interesting as well as significant to find that each case having frequent and severe attacks of migraine shows, then, *some abnormality of the stool, usually putrefaction, with an alternation of blood-pressure, and these are usually accompanied by delay of kidney function with some disturbance of nitrogen output.*

Case No.	Specific gravity, range.	Examination of												Result. Kidney function.	Blood urea. Milligrams per liter.
		Fluids.				Chlorides.				Nitrogen.					
						Day.		Night.		Day.		Night.			
		Day.	Night.	Total.	Result.	Grams.	Per cent.	Grams.	Per cent.	Grams.	Per cent.	Grams.	Per cent.		
1	23	864	170	1034	Normal	9.67	1.16	1.68	0.90	4.95	0.55	2.38	1.40	Normal	85
2	8	1448	620	2068	Poor	7.30	0.50	1.90	0.30	11.60	0.80	8.00	1.30	Poor	890
3	8	1074	670	1744	Fair	5.47	0.50	3.90	0.58	12.00	1.10	0.13	0.20	Poor	240
4	14	1140	640	1780	Fair	3.30	0.29	2.04	0.32	5.01	0.44	4.16	0.65	Poor	160
5	23	804	620	1424	Poor	5.50	0.69	1.98	0.32	5.64	0.70	6.01	0.97	Fair	120
6	17	1178	580	1758	Normal	5.30	0.44	2.61	0.45	6.60	1.70	6.50	0.90	Normal	119
7	6	902	603	1532	Poor	9.49	0.78	1.60	0.39	5.23	0.80	3.21	0.51	Fair	300
8	6	1378	550	1928	Poor	6.33	0.46	1.18	4.96	4.96	0.36	1.76	0.32	Poor	280
9	18	1086	722	1808	Normal	4.67	0.40	3.82	0.40	3.69	0.34	2.02	0.28	Normal	310
10	13	680	692	1372	Poor	4.48	0.65	3.80	0.54	3.73	0.54	4.13	0.59	Poor	230

Case No.	Renal function. Phenolsulphonephthalein test.			Gastric contents.	Feces.	Results.
	1st hour.	2d hour.	Total.			
1	18.0	8.5	26.5	Hyperacidity (mild)	Putrefaction	Improved.
2	14.0	15.5	29.5	Normal	Putrefaction	Improved.
3	21.0	16.5	37.5	Normal	Putrefaction	Improved.
4	14.0	8.0	22.0	Normal	Putrefaction	Improved.
5	22.0	17.5	39.5	Normal	Putrefaction	Improved.
6	27.5	13.5	41.0	Normal	Acid fermentation	Improved.
7	28.0	15.5	43.5	Hypersecretion	Putrefaction	Improved.
8	11.0	10.5	21.5	Normal	Putrefaction	Not improved.
9	41.0	15.5	56.5	Normal	Putrefaction	Improved.
10	14.5	12.5	27.0	Normal	Acid fermentation	Not treated.

Our own clinical observations confirm those of other investigators who find unusually high blood-pressure readings during the attack and varying blood-pressure readings in the intervals of the attack, either high or low.

We have further found that this variation of blood-pressure between attacks corresponds with the findings in the stool. For instance, a persistently low blood-pressure reading is apt to be associated with an excessively putrefactive movement, and as the stool condition is corrected, the attacks are less in frequency and severity, and some cases show improvement in pressure between attacks.

What, then, is the source of the irritation? Our next work on this will be an attempt to find the possible toxin—whether or not it is a chemical poison contained in the putrefactive stool.

We propose from now on to investigate in each case the abnormal chemical elements in the stool with reference to the blood-pressure findings and the kidney retention; and while still feeling that the problem of each case is an individual one, we are hoping that some useful suggestions for treatment in general may be derived from our further work.

The laboratory work was done in the laboratory of the Woman's Hospital of Philadelphia, under the direction of Dr. Berta O. Meine, director of the laboratory.

TWO CASES OF GAUCHER'S DISEASE IN ADULTS: A STUDY OF THE HISTOPATHOLOGY, BIOLOGY AND CHEMICAL FINDINGS.

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Up to the year 1916 only nineteen cases of Gaucher's disease had been reported in which the diagnosis was established by histological examination. An abstract of these cases may be found in a paper on the subject by Mandlebaum and Downey in the *Folia Hematologica,* February, 1916. Since that time the available literature reveals but two authentic cases. The first, reported by Dr. Frank A. Evans, May, 1916, occurred in a boy four years and eleven months of age with a history of splenic enlargement beginning at the age of two years, accompanied by frequent attacks of epistaxis. The histological examination of the spleen and other hemopoietic organs is typical of Gaucher's disease. The second case was described by Dr. Mark S. Reuben, January, 1918. The patient, a boy, aged two years, presented the clinical picture of Gaucher's disease, and an examination of the splenic pulp removed by aspiration revealed the characteristic cells. As a rule, clinicians are opposed to probatory puncture in cases of splenic hypertrophy on account of the danger of hemorrhage, but the diagnosis in suspected cases of Gaucher's disease may always be established if the typical cells are found in the aspirated fluid. This diagnostic procedure was employed for the first time in Gaucher's disease by Bernstein who found the characteristic cells just before splenectomy, the diagnosis being corroborated subsequently by histological examination of the spleen.

The cases reported by Knox, Wahl and Schmeisser in January,